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DATE: Wednesday, December 08, 2004

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		DB=USPT,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR	
☐	L2	(estrogen or estra$) same (amyloid$ adj2 $peptide)	14
☐	L1	(estrogen or estra$) same amyloid$	82

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 14 of 14 returned.

☐ 1. Document ID: US 6245756 B1

Using default format because multiple data bases are involved.

L2: Entry 1 of 14 File: USPT Jun 12, 2001

US-PAT-NO: 6245756
DOCUMENT-IDENTIFIER: US 6245756 B1

TITLE: Pharmaceutical preparations for treatment of estrogen deficiency in the central nervous system

DATE-ISSUED: June 12, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Patchev; Vladimir	Jena			DE
Oettel; Michael	Jena			DE
Schwarz; Sigfrid	Jena			DE
Thieme; Ina	Graitschen			DE
Roemer; Wolfgang	Jena			DE

US-CL-CURRENT: 514/176; 514/178, 514/179, 514/182

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 2. Document ID: US 6217860 B1

L2: Entry 2 of 14 File: USPT Apr 17, 2001

US-PAT-NO: 6217860
DOCUMENT-IDENTIFIER: US 6217860 B1

TITLE: Gene therapy for solid tumors, papillomas and warts

DATE-ISSUED: April 17, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Woo; Savio L. C.	Houston	TX		
Chen; Shu-Hsia	Houston	TX		

US-CL-CURRENT: 424/93.2; 424/93.6, 435/320.1, 514/44

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. De

☐ 3. Document ID: US 6066624 A

L2: Entry 3 of 14 File: USPT May 23, 2000

US-PAT-NO: 6066624
DOCUMENT-IDENTIFIER: US 6066624 A

TITLE: Gene therapy for solid tumors using adenoviral vectors comprising suicide genes and cytokine genes

DATE-ISSUED: May 23, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Woo; Savio L. C.	Houston	TX		
Chen; Shu-Hsia	Houston	TX		

US-CL-CURRENT: 514/44; 424/93.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. De

☐ 4. Document ID: US 6017734 A

L2: Entry 4 of 14 File: USPT Jan 25, 2000

US-PAT-NO: 6017734
DOCUMENT-IDENTIFIER: US 6017734 A

TITLE: Unique nucleotide and amino acid sequence and uses thereof

DATE-ISSUED: January 25, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Summers; Max D.	Bryan	TX		
Braunagel; Sharon C.	Bryan	TX		
Hong; Tao	Bryan	TX		

US-CL-CURRENT: 435/69.7; 435/320.1, 435/348, 435/365, 435/91.4, 536/23.1, 536/23.72, 536/24.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. De

☐ 5. Document ID: US 6010849 A

L2: Entry 5 of 14 File: USPT Jan 4, 2000

US-PAT-NO: 6010849
DOCUMENT-IDENTIFIER: US 6010849 A

TITLE: Sequence-directed DNA binding molecules compositions and methods

DATE-ISSUED: January 4, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Edwards; Cynthia A.	Menlo Park	CA		
Cantor; Charles R.	Boston	MA		
Andrews; Beth M.	Maynard	MA		
Turin; Lisa M.	Redwood City	CA		
Fry; Kirk E.	Palo Alto	CA		

US-CL-CURRENT: 435/6; 435/7.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw. D

☐ 6. Document ID: US 5869241 A

L2: Entry 6 of 14 File: USPT Feb 9, 1999

US-PAT-NO: 5869241
DOCUMENT-IDENTIFIER: US 5869241 A

TITLE: Method of determining DNA sequence preference of a DNA-binding molecule

DATE-ISSUED: February 9, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Edwards; Cynthia A.	Menlo Park	CA		
Cantor; Charles R.	Boston	MA		
Andrews; Beth M.	Maynard	MA		
Turin; Lisa M.	Redwood City	CA		
Fry; Kirk E.	Palo Alto	CA		

US-CL-CURRENT: 435/6; 435/91.1, 435/91.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw. D

☐ 7. Document ID: US 5744131 A

L2: Entry 7 of 14 File: USPT Apr 28, 1998

US-PAT-NO: 5744131
DOCUMENT-IDENTIFIER: US 5744131 A

TITLE: Sequence-directed DNA-binding molecules compositions and methods

DATE-ISSUED: April 28, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Edwards; Cynthia A.	Menlo Park	CA		
Fry; Kirk E.	Palo Alto	CA		
Cantor; Charles R.	Boston	MA		
Andrews; Beth M.	Maynard	MA		

US-CL-CURRENT: 424/78.08; 436/501, 514/1

Full | Title | Citation | Front | Review | Classification | Date | Reference | [illegible] | [illegible] | Claims | KWIC | Draw De

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☐ 8. Document ID: US 5738990 A

L2: Entry 8 of 14 File: USPT Apr 14, 1998

US-PAT-NO: 5738990
DOCUMENT-IDENTIFIER: US 5738990 A
**** See image for Certificate of Correction ****

TITLE: Sequence-directed DNA-binding molecules compositions and methods

DATE-ISSUED: April 14, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Edwards; Cynthia A.	Menlo Park	CA		
Fry; Kirk E.	Palo Alto	CA		
Cantor; Charles R.	Boston	MA		
Andrews; Beth M.	Maynard	MA		

US-CL-CURRENT: 435/6; 435/320.1, 435/69.1, 536/24.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | [illegible] | [illegible] | Claims | KWIC | Draw De

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☐ 9. Document ID: US 5726014 A

L2: Entry 9 of 14 File: USPT Mar 10, 1998

US-PAT-NO: 5726014
DOCUMENT-IDENTIFIER: US 5726014 A

TITLE: Screening assay for the detection of DNA-binding molecules

DATE-ISSUED: March 10, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Edwards; Cynthia A.	Menlo Park	CA		

Cantor; Charles R.	Boston	MA
Andrews; Beth M.	Watertown	MA
Turin; Lisa M.	Berkeley	CA

US-CL-CURRENT: 435/6; 435/91.2, 436/501

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 10. Document ID: US 5693463 A

L2: Entry 10 of 14 File: USPT Dec 2, 1997

US-PAT-NO: 5693463
DOCUMENT-IDENTIFIER: US 5693463 A

TITLE: Method of ordering sequence binding preferences of a DNA-binding molecule

DATE-ISSUED: December 2, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Edwards; Cynthia A.	Menlo Park	CA		
Fry; Kirk E.	Palo Alto	CA		
Cantor; Charles R.	Boston	MA		
Andrews; Beth M.	Maynard	MA		

US-CL-CURRENT: 435/6; 435/7.23, 536/23.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 11. Document ID: US 5631236 A

L2: Entry 11 of 14 File: USPT May 20, 1997

US-PAT-NO: 5631236
DOCUMENT-IDENTIFIER: US 5631236 A

TITLE: Gene therapy for solid tumors, using a DNA sequence encoding HSV-Tk or VZV-Tk

DATE-ISSUED: May 20, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Woo; Savio L. C.	Houston	TX		
Chen; Shu-Hsia	Houston	TX		

US-CL-CURRENT: 514/44; 424/93.6, 435/320.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 12. Document ID: US 5622981 A

L2: Entry 12 of 14 File: USPT Apr 22, 1997

US-PAT-NO: 5622981
DOCUMENT-IDENTIFIER: US 5622981 A

TITLE: Use of metabotropic receptor agonists in progressive neurodegenerative diseases

DATE-ISSUED: April 22, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Eveleth; David D.	Mission Viejo	CA		
Kelleher; Judith A.	Irvine	CA		
Cotman; Carl W.	Santa Ana	CA		

US-CL-CURRENT: 514/380; 514/561

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 13. Document ID: US 5578444 A

L2: Entry 13 of 14 File: USPT Nov 26, 1996

US-PAT-NO: 5578444
DOCUMENT-IDENTIFIER: US 5578444 A

TITLE: Sequence-directed DNA-binding molecules compositions and methods

DATE-ISSUED: November 26, 1996

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Edwards; Cynthia A.	Menlo Park	CA		
Cantor; Charles R.	Boston	MA		
Andrews; Beth M.	Maynard	MA		
Turin; Lisa M.	Redwood City	CA		
Fry; Kirk E.	Palo Alto	CA		

US-CL-CURRENT: 435/6; 435/7.23, 536/23.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 14. Document ID: WO 3102016 A2

L2: Entry 14 of 14 File: EPAB Dec 11, 2003

PUB-NO: WO003102016A2
DOCUMENT-IDENTIFIER: WO 3102016 A2
TITLE: AMYLOID PEPTIDE INACTIVATING ENZYME TO TREAT ALZHEIMER'S DISEASE

PUBN-DATE: December 11, 2003

INVENTOR-INFORMATION:

NAME	COUNTRY
HERSH, LOUIS B	US

INT-CL (IPC): C07 K 0/
EUR-CL (EPC): G01N033/50; G01N033/68

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Terms	Documents
(estrogen or estra$) same (amyloid$ adj2 $peptide)	14

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L1: Entry 13 of 82 File: USPT Nov 18, 2003

DOCUMENT-IDENTIFIER: US 6649196 B2
**** See image for Certificate of Correction ****
TITLE: Methods of reducing .beta.-amyloid polypeptides

Other Reference Publication (36):
Xu et al., "Estrogen reduces neuronal generation of Alzheimer .beta.-amyloid peptides," Nat. Med., 1998, 4(4):447-451.

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L1: Entry 18 of 82 File: USPT Feb 25, 2003

DOCUMENT-IDENTIFIER: US 6524616 B1
TITLE: COMPOSITIONS AND METHODS FOR TREATING OR PREVENTING NEURODEGENERATION AND COGNITIVE DECLINE AND DYSFUNCTION ASSOCIATED WITH ALZHEIMER'S DISEASE, AGING, OTHER DEMENTIA RELATED DISORDERS AND ESTROGEN DEFICIENCY RELATED CONDITIONS

Detailed Description Text (6):
FIG. 5 is a schematic diagram of the mechanisms of action for the compositions of the present invention. By its genomic CNS interaction with estrogen receptors in the brain (hypothalamus; cerebral cortex; limbic system and hippocampus) and additional on-genomic activity, the following estrogen-influenced positive CNS events can be emonstrated: A. upgrade in the synthesis and activity of ChAT, BDNGF and other associated factors; B. stimulation of dendritic growth; C. estrogen reduces the deposition of .beta.-amyloid; D. improvements in cerebral and cerebellar blood flow and vasodilatory response to acetylcholine in women with cerebrovascular disease; E. improvement in serotonin biochemistry, synthesis and release; and F. a CNS anti-oxidant.

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L1: Entry 38 of 82 File: USPT Dec 25, 2001

DOCUMENT-IDENTIFIER: US 6333317 B1
TITLE: Regulation of amyloid precursor protein (APP) expression by administration of an estrogenic compound

Brief Summary Text (5):
Several lines of studies suggest that postmenopausal women with lower levels of endogenous estrogen may be predisposed to the development of AD. Studies in experimental animal models provide a convincing rational for the role of estrogen replacement therapy and prevention of dementia. See, for example, Birge, J. Am. Geriatric. Soc. 44, 865, (1996). These studies suggest that estrogen deficiency in postmenopausal women apparently increases their susceptibility to the neurodegenerative changes of aging and AD, and that this risk can be decreased by estrogenic replacement therapy. Peganini et al. Am. J. Epidemiol, 140, 256 (1994). Estrogen treatment of cell culture reportedly promotes non-amyloidogenic APP processing and soluble APP (APPs) secretion. Jaffe et al., J. Biol. Chem. 269, 13065 (1994).

Brief Summary Text (17):
Estrogen receptors within the brain have a regional distribution strikingly similar to that characteristics of AD-type brain pathology, Thomlinson, B. E.(1992): In Greenfield's neuropathology (Adams, J. H. and Duchen L. W. eds)pp 1284-1410 Oxford University Press. In addition recent findings suggest that estrogen replacement therapy my be protective against AD, Henderson, et al., Arch neurol (1994). In order to test the hypothesis that gonadal esteroids might play a role in regulating APP metabolism, Jaffe, et al., J. Biol. chem.269, 13065 (1994) investigated the possible effect of estrogen on the metabolism of Alzheimer's amyloid precursor protein. Using a cell line that contains high level of estrogen receptors, these authors found that the treatment with 17.beta.-estradiol is associated with the accumulation of APPs in the medium, indicative of non-amyloidogenic processing. However, these authors found no changes in the levels of intracellular immature or mature APP holoproteins, suggesting that estrogen may increase the secretory metabolism of APP.

Brief Summary Text (26):
Another object of this invention is to provide a method of determining the capacity of a drug to modulate expression, production, or formation of amyloid precursor protein (APP) in an eukaryotic cell comprising, contacting a drug with an eukaryotic cell containing estrogen receptors.

Detailed Description Text (6):
Jaffe et al. J. Biolo. Chem.269, 13065 (1994) disclose the possible effect of estrogen on the metabolism of the Alzheimer's amyloid precursor protein (APP). Using the breast carcinoma cell line, these authors found that treatment with physiological concentrations of 17.beta.-estradiol is associated with extracellular accumulation of soluble APP (APPs). However, these authors found no changes in the levels of intracellular immature or mature APP holoproteins, suggesting that estrogen may increase the secretory metabolism of APP.

Detailed Description Text (10):
Previously, we have shown that stimulation of certain cell-surface receptors

coupled to cAMP formation in astrocytes increases the production of APP mRNA and APP holoprotein. It is suggested that the up regulation or aberrant activation of certain receptors in brain regions that are vulnerable to damage can stimulate APP overexpression in brain cells and, thereby, contribute to amyloid production. Because APP over expression can cause neurodegeneration and cognitive dysfunction, the inventors have shown that such substances as neuroactive steroids and related products, e.g., estrone and 17.beta.-estradiol and the like, are promising drug candidates for the treatment of Alzheimer's, Parkinson's, Lou Gehrig's Disease (amylotrophic lateral sclerosis), multiple sclerosis, ischemia, traumatic brain injury, epileptic seizure, and the like, which may have their roots in the formation or presence of amyloid plaques.

Detailed Description Text (14):
Thus, the present invention is directed to a method of modulating the expression, production, or formation of amyloid precursor protein (APP) in a subject comprising administering to the subject an effective amount of a lipophilic hormone, such as estrone or 17.beta.-pestradiol, an analog of a lipophilic hormone, a substance that is a ligand, an agonist, or an antagonist of a receptor that is coupled to a lipophilic hormone, or a compound that regulates the activity of cytosolic or nuclear receptors. In specific embodiments of the invention the lipophilic hormones and related compounds comprise; Allopregnanolone; Allotetrahydrodeoxycorticosterone; Alphaxalone; Androsterone, 4-Androstane-3,17-dione, Corticosterone; Corticosterone: HBC complex; Danazol; Dehydroepiandrosterone sulfate sodium; Dexamethasone; 17.beta.-Estradiol; 17.beta.-Estradiol: HBC complex; Estrone; Etiocholanolone; FGIN I-27; Fluoxymesterone; Hydrocortisone; Hydrocortisone: HBC complex; Methandrostenolone; Nandrolone decanoate; Oxandrolone; Oxymetholone; Prednisolone; Pregnenolone sulfate sodium; Progesterone; Progesterone: HBC complex; Spironolactone; Stanolone; Tamoxifen; 3-hydroxy; citrate; (E)-; Tamoxifen, 4-hydroxy-, (E)-; Tamoxifen, 4-hydroxy-, (Z)-; Testosterone: HBC complex; Testosterone, 17.beta.-cypionate; Testosterone, 17.beta.-decanoate; Testosterone, 17.beta.-heptanoate; Testosterone, 17.beta.-isocapronate; Testosterone, 17.alpha.-methyl-; Testosterone, 17.beta.-propionate; Triamcinolope; U-73122; U-73343; Endosulfan; dieldrin; methoxychlor; thyroid hormones; nethimazole (antithyroid hormones); human growth hormones; gonadotropin; vasopressin; calcitonin; adrenal cortical hormones; insulin; anabolic steroids; and antineoplastics.

Detailed Description Text (19):
It is particularly advantageous to treat the subject in need by administering an effective amount of a lipophilic hormone such as estrone or 17.beta.-estradiol. It is noteworthy that the present invention is also directed to a method of determining the capacity of a drug to inhibit the expression, production, or formation of amyloid precursor protein (APP) in a cell comprising contacting an estrogenic drug, an analog of an estrogenic drug, a substance that is a ligand, an agonist, or an antagonist of an estrogen receptor, with a cell culture that has the capacity to synthesize APP holoprotein. The level of APP mRNA or holoprotein produced from the cell culture in the presence of the drug is then compared with the level of APP mRNA or holoprotein produced from the cell culture in the absence of the drug. The cell can be any type of microbial, plant, or animal cell, so long as the cell has the capacity to express, produce, or otherwise form APP. The cell is preferably an eukaryotic cell. More preferably, the eukaryotic cell can further be a yeast cell, insect cell, invertebrate, vertebrate, or mammalian, including animal or human.

Detailed Description Text (20):
It should be apparent that the present invention is directed to a method of alleviating the negative effects of a neurological disorder or neurodegenerative disease stemming from the aberrant expression, production, or formation of amyloid precursor protein (APP) in a subject. In a particular embodiment, an effective amount of a lipophilic hormone such as estrone or 17.beta.-estradiol, or an analog

or agonist thereof, is administered to the subject suffering from the disorder or disease. As described herein, the present method of modulating amyloid precursor protein (APP) expression in a subject may also comprise administering to the subject an effective amount of a substance that regulates APP promoter activity, either by stimulating APP promoter activity or retarding it.

Detailed Description Text (22):
Moreover, compositions for modulating the expression, production, or formation of amyloid precursor protein (APP) in a subject are intended which comprise neuroactive steroids, such as estrone or 17.beta.-estradiol, analogs of neuroactive steroids, a substance that is a ligand, an agonist, or an antagonist of a receptor that is coupled to a neuroactive steroid, or a compound that regulates the activity of cytosolic or nuclear receptors.

Detailed Description Text (27):
By a "therapeutically effective amount" or simply "effective amount" of an active compound, such as an analog of estrogen, is meant a sufficient amount of the compound to treat or alleviate the negative effects of a neurological disorder or neurodegenerative disease stemming from an increase in the level of expression, production, or formation of amyloid precursor protein (APP) at a reasonable benefit/risk ratio applicable to any medical treatment. It will be understood, however, that the total daily usage of the active compounds and compositions of the present invention will be decided by the attending physician within the scope of sound medical judgment. The specific therapeutically effective dose level for any particular patient will depend upon a variety of factors including the disorder being treated and the severity of the disorder; activity of the specific compound employed; the specific composition employed; the age, body weight, general health, sex and diet of the patient; the time of administration, route of administration, and rate of excretion of the specific compound employed; the duration of the treatment; drugs used in combination or coinciding with the specific compound employed; and like factors well known in the medical arts.

Other Reference Publication (3):
Jaffe et al., "Estrogen Regulates Metabolism of Alzheimer Amyloid .beta. Precursor Protein," J. Biological Chemistry, 269(18), 13065-13068 (May 6, 1994).*

CLAIMS:

1. A method of inhibiting expression, production, or formation of amyloid precursor protein (APP) in a subject comprising administering to the subject an effective amount of an agent wherein said agent is a lipophilic hormone, anaalog of a lipophilic hormone, a substance that is a ligand, an agonist, or an antagonist of a receptor that is coupled to a lipophilic hormone, and a pharmaceutically acceptable carrier or diluent, in which the effective amount is at least about 0.20 mg/kg body weight, and wherein said agent is selected from the group consisting of androsterone; 4-androstane-3,17-dione; danazol; dehydroepiandrosterone sulfate sodium; estradiol; 17.beta.-estradiol; 17.beta.-estradiol; HBC complex; estrone; fluoxymesterone; methandrostenolone; nandrolone decanoate; oxandrolone; oxymetholone; pregnenolone sulfate sodium; progesterone; progesterone; HBC complex; stanolone; testosterone; HBC complex; testosterone, 17.beta.-cypionate; testosterone, 17.beta.-decanoate; testosterone, 17.beta.-heptanoate; testosterone, 17.beta.-isocapronate; testosterone, 17.alpha.-methyl-; testosterone, 17.beta.-propianate; methoxychlor; and gonadotropin.

9. A method of alleviating the negative effects of a neurological disorder or neurodegenerative disease stemming from the abberant expression, production, formation, or overexpression of amyloid precursor protein (APP) in a subject, comprising administering to a subject suffering from said disorder or disease an effective amount of an agent wherein said agent is a lipophilic hormone, an analog of a lipophilic hormone, a substance that is a ligand, an agonist, or an antagonist

of a receptor that is coupled to a lipophilic hormone, and a pharmaceutically acceptable carrier or diluent, in which the effective amount is at least about 0.20 mg/kg body weight, and wherein said agent is selected from the group consisting of androsterone; 4-androstane-3,17-dione; danazol; dehydroepiandrosterone sulfate sodium; estradiol; 17.beta.-estradiol; 17.beta.-estradiol; HBC complex; estrone; fluoxymesterone; methandrostenolone; nandrolone; decanoate; oxandrolone; oxymetholone; pregnenolone sulfate sodium; progesterone; progesterone; HBC complex; stanolone; testosterone; HBC complex; testosterone, 17.beta.-cypionate; testosterone, 17.beta.-decanoate; testosterone, 17.beta.-heptanoate; testosterone, 17.beta.-isocapronate; testosterone, 17.alpha.-methyl-; testosterone, 17.beta.-propionate; methoxychlor; and gonadotropin.

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L1: Entry 45 of 82 File: USPT Jun 12, 2001

DOCUMENT-IDENTIFIER: US 6245756 B1
TITLE: Pharmaceutical preparations for treatment of estrogen deficiency in the central nervous system

Brief Summary Text (18):
to increase the resistance of nerve cells against pathological action (Y. Goodman, et al, "Estrogens Attenuate and Corticosterone Exacerbates Excitotoxicity, Oxidative Injury, and Amyloid b-peptide Toxicity in Hippocampal Neurons", J. Neurochem. 66, 1836 to 1844, 1996).

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L1: Entry 50 of 82 File: USPT Jun 27, 2000

DOCUMENT-IDENTIFIER: US 6080778 A
TITLE: Methods for decreasing beta amyloid protein

Abstract Text (1):
Blood cholesterol levels are correlated with production of amyloid .beta. protein (A.beta.), and are predictors of populations at risk of developing AD. Methods for lowering blood cholesterol levels can be used to decrease production of A.beta., thereby decreasing the risk of developing AD. The same methods and compositions can also be used for treating individuals diagnosed with AD. Methods include administration of compounds which increase uptake of cholesterol by the liver, such as the administration of HMG CoA reductase inhibitors, administration of compounds which block endogenous cholesterol production, such as administration of HMG CoA reductase inhibitors, administration of compositions which prevent uptake of dietary cholesterol, and administration of combinations of any of these which are effective to lower blood cholesterol levels. Methods have also been developed to predict populations at risk, based on the role of cholesterol in production of A.beta.. For example, individuals with Apo E4 and high cholesterol, defined as a blood cholesterol level of greater than 200 mg/dl, post menopausal women with high cholesterol levels--especially those who are not taking estrogen, or individuals which high blood cholesterol levels who are not obese are all at risk of developing AD if blood cholesterol levels are not decreased.

Brief Summary Text (8):
Blood cholesterol levels are correlated with production of amyloid .beta. protein (A.beta.), and are predictors of populations at risk of developing AD. Methods for lowering blood cholesterol levels can be used to decrease production of A.beta., thereby decreasing the risk of developing AD. The same methods and compositions can also be used for treating individuals diagnosed with AD. Methods include administration of compounds which increase uptake of cholesterol by the liver, such as the administration of HMG CoA reductase inhibitors, administration of compounds which block endogenous cholesterol production, such as administration of HMG CoA reductase inhibitors, administration of compositions which prevent uptake of dietary cholesterol, and administration of combinations of any of these which are effective to lower blood cholesterol levels. Methods have also been developed to predict populations at risk, based on the role of cholesterol in production of A.beta.. For example, individuals with Apo E4 and high cholesterol, defined as a blood cholesterol level of greater than 200 mg/dl, post menopausal women with high cholesterol levels--especially those who are not taking estrogen, or individuals which high blood cholesterol levels who are not obese are all at risk of developing AD if blood cholesterol levels are not decreased. In the preferred embodiment, individuals with these risk factors are treated to lower blood cholesterol levels prior to developing any mental impairment attributable to AD, based on accepted neuropsychiatric and diagnostic criteria in clinical practice. Treatment is based on adminstration of one or more compositions effective to lower cholesterol blood levels at least 10%, which is believed to be sufficient to decrease production of A.beta..

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L1: Entry 76 of 82 File: EPAB Sep 30, 1999

DOCUMENT-IDENTIFIER: WO 9948488 A2
TITLE: METHODS FOR DECREASING BETA AMYLOID PROTEIN

Abstract Text (1):
CHG DATE=19991102 STATUS=O>Blood cholesterol levels are correlated with production of amyloid beta protein ((A) beta), and are predictors of populations at risk of developing AD. Methods for lowering blood cholesterol levels can be used to decrease production of A beta , thereby decreasing the risk of developing AD. The same methods and compositions can also be used for treating individuals diagnosed with AD. Methods include administration of compounds which increase uptake of cholesterol by the liver, such as the administration of HMG CoA reductase inhibitors, administration of compounds which block endogenous cholesterol production, such as the administration of HMG CoA reductase inhibitors, administration of compositions which prevent uptake of dietary cholesterol, and administration of combinations of any of these which are effective to lower blood cholesterol levels. Methods have also been developed to predict populations at risk, based on the role of cholesterol in production of A beta . For example, individuals with Apo E4 and high cholesterol, defined as a blood cholesterol level of greater than 200 mg/dl, post menopausal women with high cholesterol levels - especially those who are not taking estrogen, or individuals with high blood cholesterol levels who are not obese are all at risk of developing AD if blood cholesterol levels are not decreased.

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DATE: Wednesday, December 08, 2004

Hide?	Set Name	Query	Hit Count
		DB=USPT,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR	
☐	L2	(estrogen or estra$) adj10 (amyloidosis or alzheimer$)	115
☐	L1	(estrogen or estra$) same (amyloidosis or alzheimer$)	426

END OF SEARCH HISTORY

L2: Entry 64 of 115 File: USPT Feb 17, 1998

DOCUMENT-IDENTIFIER: US 5719137 A
TITLE: Method of preventing neurodegeneration and cognitive dysfunction using 17.alpha.-dihydroequilenin

Abstract Text (1):
A method of using a steroidal compound, 17.alpha.-dihydroequilenin, to prevent and treat neurodegeneration and cognitive dysfunction in estrogen deficient females and to reduce the risk of Alzheimer's related dementia and other senile dementia related conditions in both males and females. The method comprises administering 17.alpha.-dihydroequilenin in a therapeutically effective amount to a mammal in need of increased cognitive function or to a mammal susceptible to estrogen deficiency-related neurodegeneration or to senile dementia of the Alzheimer's type.

Brief Summary Text (2):
The present invention relates to a method of using a steroidal compound to prevent the degeneration of neurons associated with cognitive functions like memory and attention in mammals. More particularly, the present invention relates to a method of using 17.alpha.-dihydroequilenin to prevent neurodegeneration and cognitive dysfunction in estrogen deficient females and to reduce the risk of Alzheimer's related dementia in both males and females.

Brief Summary Text (16):
The use of 17.alpha.-dihydroequilenin to prevent and/or treat neurodegeneration associated with cognitive dysfunction in estrogen deficient mammals and to reduce the risk of senile dementia of the Alzheimer's type provides distinct advantages over traditional estrogen replacement therapies. 17.alpha.-dihydroequilenin has demonstrated beneficial effects on the central nervous system function without uterotrophic effects of the type associated with estradiol. In contrast to other estrogens like estradiol which are known to cause a thickening of the uterine lining and increase uterine weight, studies have shown that 17.alpha.-dihydroequilenin has minimal to no estrogenic activity in the uterus or the hypothalamic pituitary portions of the gonadal axis as determined with the following traditional measures of estrogenic potency: 1) by relative binding affinity to the human endometrial and rat uterine cytosol and nuclear estradiol receptors; 2) the Allen-Doisy test (the amount of a particular estrogen needed to double the weight of a rat uterus); 3) by its uterotrophic potency; and 4) its inability to suppress urinary gonadotropin levels in oophorectomized women [Stern, Michael, Maturitas, 4:333 (1982); Howard et al., Arch. Int. Med., 128:229 (1971)].

Detailed Description Text (2):
In the studies of the present invention using ovariectomized rats, the effects of short-term (2 to 3 days) oral 17.beta.-estradiol, subcutaneous estradiol benzoate, and oral 17.alpha.-dihydroequilenin treatment were compared versus untreated controls on the apical dendrite spine density of pyramidal cells of the CA1 region of the hippocampus (n=4 brains/group). All three treatments resulted in increased spine densities relative to untreated controls, and there were no apparent differences between the treatments. These results suggest that 17.alpha.-dihydroequilenin is a prime candidate for a single-agent hormone replacement therapy to treat mammals with an estrogen deficiency condition such as menopause as

well as to reduce the chronic disease risk of Alzheimer's dementia or other dementia related conditions in both males and females.

Other Reference Publication (3):
The Potential Role For Estrogen Replacement Therapy in the Treatment of the Congnitive Decline and Neurodegeneration Associated With Alzheimer's Disease Neurogiology of Aging, vol. 15, Suppl. 2, pp. S195-S197, 1994.

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L2: Entry 65 of 115 File: USPT Nov 11, 1997

DOCUMENT-IDENTIFIER: US 5686476 A
TITLE: Methods of inhibiting Alzheimer's Disease

Other Reference Publication (23):
"Women on Estrogen Appear at Less Risk of Alzheimer's", Indianapolis Star, Nov. 10, 1993.

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L2: Entry 66 of 115 File: USPT Jul 29, 1997

US-PAT-NO: 5652259
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INVENTOR-INFORMATION:

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US-CL-CURRENT: 514/422; 514/317, 514/319, 514/324, 514/443

CLAIMS:

We claim:

1. A method of increasing TGF-.beta. expression in the brain comprising administering to a human in need thereof an effective amount of a compound of Formula I ##STR6## wherein R.sup.1 and R.sup.3 are independently hydrogen, ##STR7## wherein Ar is optionally substituted phenyl; R.sup.2 is pyrrolidine; or a pharmaceutically acceptable salt of solvate thereof.

2. The method of claim 1 wherein said administration is prophylactic.

3. The method of claim 1 wherein said human is a post-menopausal female.

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